Poore (C. T.)

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS,

WITH AN

ANALYSIS OF CASES.

BY

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Complimentary of C. T. Poore

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS,

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PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS, WITH AN ANALYSIS OF CASES.

The affection of the muscular system designated "pseudo-hypertrophic," or "myosclerotic" paralysis, which may be defined as a diminution of motility with increase in the volume of the muscles, was first described by the Italian physicians Coste and Gioja, in 1838, who mention two cases under their care at the Hospital for Incurables at Naples. In 1852 Edward Meryon reported four cases, in the "Transactions of the Medico-Chirurgical Society of London;" but it was not until 1861 that this disease was carefully studied and brought before the profession, by Duchenne, in his work on electricity; and again by the same author in 1868, in a series of articles, with an analysis of thirteen cases (*Archives Générales de Médecine*), from which all writers on this disease have largely drawn when treating of this complaint.

Since then numerous cases have been reported in the German, French, and English medical journals.

In 1868 Dr. Clymer published an article in the appendix to Aitken's "Science and Practice of Medicine," and again in 1870 a lecture by the same author appeared in the *Medical Record* for July 15th, with a review of the cases up to that date. In November of the same year, Drs. Ingalls and Webber, of Boston, published the first case described in American medical literature, with a partial analysis of forty-one cases, in the *Boston Medical and Surgical Journal*. Since then cases have been reported by Dr. William Pepper (*Philadelphia Medical Times*, June and July, 1871); Dr. S. Weir Mitchell (*Photographic Review*, October, 1871); Dr. E. M. Estrazulas (Annual Supplement to *Obstetrical Journal of*

Great Britain, etc., September, 1873); Dr. C. H. Drake (*Philadelphia Medical Times*, August 29, 1874).

From the above it would seem that the disease is quite rare; but cases may have escaped notice, not having been recognized, or having been considered as cases of progressive muscular atrophy, a disease which the one under consideration resembles in many of its symptoms. I am informed that a case was brought to an orthopœdic dispensary in this city in 1869, but not at the time recognized, and from the history there were probably three cases occurring in the same family, in Vermont.

As a further contribution to the literature of this subject the following case is presented, with an analysis of eighty-five cases collect fro various American and foreign medical journals. For valuable assistance in preparing this paper I am under great obligations to Drs. C. S. Bull, Edward Frankel, and George R. Cutter, the latter for looking over the Swedish medical literature.

E. G., nine years old, was a perfectly healthy and medium-sized child at birth. Her parents are still alive; her mother is healthy, but has been subject to severe headaches from puberty. Her father is intemperate. Her maternal grandmother died of cancer of the breast, and her grandfather of some disease attendant on old age. Her mother has never heard of any member of her husband's family who has been paralyzed or deformed. Patient's mother has had eight children, five of whom she has lost, two infants at birth, one two years old of typhoid fever, one at five of bilious fever, and one killed by an accident. There are three daughters alive and perfectly healthy; none of her children have ever had convulsions. Patient began to walk when two years old, and walked well until her fourth year, when it was noticed that in running she dragged her toes on the ground, and seemed weak in her legs. She fell very often; the least thing would throw her down. When five years old it was noticed that the muscles on the back of her legs were shortened. Difficulty in walking has gradually increased ever since. Until her third year she was sickly, had chronic diarrhœa, and at that age was small for her years, but since then has grown rapidly,

but no portion of her body has increased more than another.

Last winter her feet and legs began to feel cold and look purple; this condition increased during the summer. She has had no children's disease except mumps. Has never complained of any pain in her legs. She has had at times difficulty in holding her water. All her brothers and sisters walked well.

The patient was admitted to St. Mary's Free Hospital for Children October 7, 1874. The following was her condition at the time of her admission:

She is a heavy-looking child, rather thick-set, forty-six inches in height, healthy in appearance; sensation, as far as can be ascertained, normal; in intelligence she is dull and stupid. Heart and lungs are normal. The muscles all over her body seem enlarged, so as to give her an unwieldy appearance. The muscles of her calves are well developed, and when they are contracted are hard, and stand out prominently, but when relaxed are hard to define; they seem covered by a thick layer of fat. The thighs are well developed, but the muscles do not feel as hard as those on the posterior aspect of leg. The glutei are soft even when contracted. The extensor muscles of the spine, in the lumbar region, are strongly developed, and stand out like hard bands when in action. Muscles of the arm and forearm are firm when contracted, but are not increased in size, and cannot withstand much resistance.

The muscles on the anterior aspect of leg seem covered with a deep layer of fat, and cannot be defined. Vasti interni appear large, but are soft and flabby. Her feet are in a position resembling talipes equinus, with a slight tendency to varus, being extended at the ankle-joint. There is a claw-like bending of the toes.

Passive movements of the foot on the ankle joint, both flexion and extension, can be made without difficulty; but active movements are impossible. When in bed she can flex the thigh on the pelvis, and the leg on the thigh—the right better than the left—but not with much energy.

Grasp of hand weak. Can dress and undress herself.

When standing, there is a marked lordosis in the lumbar region. She stands with her legs widely separated, and the upper portion of her body and shoulders thrown back.

In walking, she swings her body from side to side in a waddling manner. In progression, the anterior portion of her feet only is in contact with the ground. When she first starts off to walk, her heel is almost flat on the floor, but, on taking one or two steps, the gastrocnemii are seen to contract more and more powerfully, raising the heel more and more from the floor, with a hastening gait which ends by her falling or catching some firm object.

Skin of feet and legs red and mottled, easily chilled, and then becomes purple; the same is true of her hands, arms, and body.

No pain on pressure along the spine.

When she attempts to sit down on the floor, she drops down; on rising, has to draw herself up by climbing hand over hand, seizing some firm object for that purpose.

Turning from the prone to the supine position when in bed is difficult.

She cannot cross one leg over the other when in a sitting position.

The strength of the gastrocnemii is considerable; that of the arm and forearm weak.

Electro-muscular contractility is good in all muscles except those on anterior aspect of legs.

When she attempts to lean forward she falls, unless she is supported.

Has no trouble with her bladder or rectum.

Her appetite is good.

Reëxamination April 17, 1875.

Since her admission into the hospital her back and muscles have been daily galvanized. She has had good, nourishing diet, and has taken cod-liver oil; but there has been no stay in the progress of the disease. Her difficulty in walking has gradually increased, and her falls have been more frequent. For some time she has been unable to dress and undress herself. She has lost much of the fullness about her face, and the muscles of her arm and forearm are more flabby, and do not

contract with as much power as at the time of the first examination.

The sacro-lumbar muscles have decreased much in size, and with this she has had more difficulty in keeping her balance. Her gastrocnemii are still hard when contracted, but have diminished somewhat in size. The mottling of the skin is more marked. All her movements are executed in a more clumsy manner, and she is not inclined to move about much. I cannot satisfy myself that there has been any change in the electro-muscular contractility, except in the lumbar muscles, where it is diminished.

The disease is divided into three, and by some writers into four stages, viz., that of weakness of the limbs, hypertrophy, a stationary period, and finally one of paralysis and atrophy.

In all cases, weakness of the lower extremities marks the beginning of the disease. There is no fever, and in most cases no pain, nor any neurotic symptom. If the disease begins in early infancy, before the period of walking usually commences, this may be delayed, or the little patient may always use her limbs in a very clumsy manner, or may never walk at all.

If, on the other hand, the disease is later in manifesting itself, the only symptoms to attract attention at first are, the peculiar unsteady gait, easy fatigue, and frequent falls of the patient, especially in running.

In eighty-five cases, three never walked, twenty-four never walked well, one is reported as coming on gradually, fifty-two walked well at first, and in five cases no mention is made of the period of walking.

Of those who never walked well, two began to walk at eighteen months, three at two years, three at two and one-half years, two at three years, one at three and one-half years, four at four years, one at five years, and five are reported as walking late and badly.

Of the fifty-two patients who experienced no difficulty in walking at first, in thirty-five the date of walking is not reported. In the fifteen cases in which it is mentioned, the earliest date of walking is one year; the latest, three years. Four did not walk until their second year; three walked early—one at fifteen months, one at eighteen months.

Again, of the fifty-two patients who walked well at first, difficulty in walking began at—

2½ years	2 cases.	11 years	3 cases.
3 "	2 "	16 "	3 "
3½ "	1 case.	24 "	1 case.
4 "	2 cases.	26 "	1 "
5 "	8 "	28 "	1 "
6 "	6 "	32 "	1 "
7 "	9 "	37 "	1 "
8 "	2 "	40 " (about)	1 "
9 "	1 case.	Childhood	1 "
10 "	5 cases.	Early age	1 "

Thus, in eighty-five cases, it is found that in thirty-eight the disease first manifested itself before the age of five years, in twenty-six between the fifth and tenth year, in six between the tenth and sixteenth year, and in only six after that age, namely: one at twenty-four, one at twenty-six, one at twenty-eight, one at thirty-two, one at thirty-seven, and one about forty.

Out of the twenty-six patients in whom the disease developed itself before they walked, the calves were always enlarged in three, and when two of these came under observation, in their thirteenth year, they had never walked.

The remaining twenty-three did not begin to use their lower limbs until a late period, and even in those who at first seemed to exhibit no characteristic difficulty in locomotion, but who in later years developed the disease, the same backwardness in walking was often noticed.

In two cases the disease began with convulsions; one is reported by Duchenne (*Archiv. Gén. de Méd.*, 1868) in a boy who was well-formed and had walked well until his fifth year, when he was seized with convulsions, lasting some hours, unaccompanied by any fever, followed by weakness in his lower limbs. After some months a progressive increase in the size of the calves and lumbar muscles was noticed, and in his eighth year he showed all the symptoms of an advanced case of pseudo-hypertrophic paralysis. The other case, reported by Dr. C. H. Drake (*Philadelphia Medical Times*, August 27, 1874), was a boy who had lost two brothers from "brain-fever," and was seized with the same disease, accompanied with violent and long-continued convulsions; he has never been

well since. One year later difficulty in walking was noticed, followed by a gradual increase in the size of the muscles of the calves, forearm, and in other parts of his body, and at twelve years of age he exhibited all the symptoms of the disease under consideration.

One case commenced with pain in the lower limbs (Seidel, *Centralblatt*, 1867, p. 666).

One, a man of twenty-six, who had done a great amount of walking, experienced a feeling of stiffness in walking, and rheumatic pains on the outside of his thigh; three months later the muscles of his thighs began to enlarge, followed in three weeks by those of his calves (Brown, *Edinburgh Medical Journal*, vol. xv., 1870, p. 1079). One patient complained that his legs ached after using them (Russell, *Medical Times and Gazette*, May, 1869, p. 571). One, pain in his back at the beginning of the disease (Down, "Trans. Path. Soc.," London, 1870, p. 29). One, acute pain at times in his calves (Drake, *loc. cit.*). One, constant pain in his legs, from the beginning of the disease until he was confined to his bed (Heller, *Archiv. f. klin. Med.*, I., vi., p. 616). One had pain in the spinal column, and afterward in the lower limbs (Rakowac, *Wiener medicinisch Wochenschrift*, No. 12, 1872). One, pain between the shoulders after using her arms (Brunniche, *Hospitals-Tidende*, April 29, 1874). But all these seem to be exceptions to the rule that the disease is unaccompanied by pain. Later on, when the muscles have become greatly increased in size, there is often a feeling of tension after exertion.

The difficulty in walking, experienced at the beginning of the disease, is due to a progressive loss of power in the muscles of the legs, buttocks, and back. Those affected with the disease stand and walk with their feet widely separated, in order to give them a wider base of support. The lumbar curve is exaggerated, the upper portion of the body thrown backward, and the abdomen protruded in order to enable them to keep their balance.

Their mode of walking, even at an early period of the disease, is peculiar. With the feet widely separated, they throw their entire weight on one leg, with a lateral bending of the lumbar spine to that side; and then, with a jerk or hitch,

swing the other leg, carrying the foot through the arc of a circle, with the toes pointing downward to a position in advance of the other. This manœuvre is repeated in advancing the other foot. This alternate balancing of the body gives these patients a very peculiar and characteristic gait. This waddling, oscillating manner of walking is mentioned in fifty-eight of the eighty-five cases; and in twenty-seven no mention is made of the mode of walking. The incurvation of the lumbar spine in the erect position, which Duchenne considers one of the constant symptoms of the disease, is mentioned as existing in forty-nine cases. No mention is made of it in thirty-five cases; but of these, eight did not walk at the time of observation, and two had never been able to use their lower limbs, so that, out of the eighty-five cases, all but twenty-seven are accounted for.

Duchenne calls attention to a peculiarity in the lordosis of pseudo-hypertrophic paralysis as differing from that found in atrophy, or paralysis of the abdominal muscles; in the latter a plumb line, let fall from the most prominent spinal process, will pass through the sacrum, while in the disease under consideration it will pass behind the plane of that bone. The period in the course of the disease at which this symptom is found is stated in only six cases: in two it was among the earliest; in one case it was seen when the child first began to walk; in one five years, and in one six years after the waddling gait was noticed—and in one case it seems to have been the first symptom.

This lordosis is due to a weakness of the extensor muscles of the spine. The manner in which these patients pull themselves up, as it were, from a sitting posture, or in getting up from the floor, shows how little strength there is in these muscles.

The difficulty which they experience in keeping their balance while standing or walking, even in the early stage of the disease, is due to the same cause. Any unevenness of the ground, any little jostle, or sudden attempt to turn, is sufficient to throw them down. This, together with the waddling gait and clumsy mode of getting up, may be, and often is, the first symptom to attract attention.

The disease is much more common among males than females: thus, in eighty-five cases, seventy-three occurred among males, and only eleven among females. In one case no mention is made of the sex.

The symptom marking the second stage of the disease, which is the most striking, and at the same time most characteristic, is the painless increase in the size of a limb, or certain muscles of a limb, from which the disease derives one of its names.

There is no rule as to the period in the disease when this hypertrophy begins.

It is impossible to give in any statistics the period from which this increase in the size of the muscles dates. But few cases have been under observation at a sufficiently early date of the trouble, and the statement of the friends of the patient, or the patients themselves, when old enough, must necessarily be inaccurate, as their attention would not be called to the condition of the muscles until they had undergone considerble change. The following table is only approximative:

In forty cases no mention is made of the time when the hypertrophy commenced.

In three cases the calves were always enlarged.

In eleven cases the enlargement of the calves was noticed at the time the difficulty in walking began.

One case dates from infancy.

In five, the calves are said to have commenced to enlarge six months after the waddling gait was noticed; in two, three months after; in three, one year after; in one, one year and a half after; in three, two years after; in two, three years after; in four, four years after; in one seven years after; and in one case there was no hypertrophy.

In the three cases in which the calves were always large—in two (Duchenne, *loc. cit.*; Griesinger, *Arch. d. Heilkunde*, vi., 1, p. 1, 1865; Schmidt's "Jahrb.," 1865, iv., p. 179) the patients never walked; in the third (Hillier, "Diseases of Children," p. 264) the child walked at twenty-one months, but always unsteadily. After his third year, his walking powers gradually declined, and at ten he was unable to raise himself in bed.

The case in which there was no increase in the size of the muscles is reported by Russell (*loc. cit.*). The muscles of his calves were hard when contracted, but not enlarged. He had not been able to stand for eighteen months; and the pathological condition of the muscular tissue, taken from the calves, was similar to the same tissue taken from his brother, who shared all the marked symptoms of pseudo-hypertrophic paralysis.

It is a question whether the disease ever begins during intra-uterine life. Hillier, Duchenne, Griesinger (*loc. cit.*), each reports a case where the calves are said to have been enlarged at birth.

The statement of some observers would indicate that the hypertrophied muscles were weakened; this is true in the later periods of the disease. Russell (*loc. cit.*), Pepper (*Philadelphia Medical Times*, June 15, 1871), B. Foster ("Clinical Medicine," London, 1874), Davidson (*Glasgow Medical Journal*, May, 1872), and others, state that the enlarged muscles are at first increased in strength. In my own case there is certainly no decrease in the power of the gastrocnemii, although the muscles in other parts of the body are weakened. Duchenne states that, from his own observation, the degree of paralysis is not in direct relation to that of the hypertrophy. Davidson considers the hypertrophy as compensatory, and explains the localization of the enlargement in the calves by the fact that they have the greatest amount of work to do in keeping the body erect, and in walking.

In those cases where most of the muscles of the body have undergone this change, the appearance of the patient would indicate great strength and endurance; while, in truth, it is with the greatest difficulty that he can walk across a room. Again, the difficulty in walking increases, notwithstanding the hypertrophy. The enlarged muscles stand out prominently, and are hard to the feel when contracted, but when relaxed are flabby, and hard to define. In some cases they are covered by a thick layer of subcutaneous fat, which dips down at times between the muscles (Dahlerup, *Nord. Med. Ark.*, vol. iv., No. 7, 1872). The skin may be so stretched over the muscles, being distended by the fat, that it cannot be pinched up.

In two cases a stage of atrophy preceded that of hypertrophy (Pepper, *loc. cit.*; Barth, *Arch. d. Heilk.*, 1871, vol. ii.). In the one case the atrophy attacked first the muscles of the calves, then those of the thigh, back, and shoulders; two years later, after the atrophy had become marked, hypertrophy began, first in the muscles of the calves, which grew until they acquired a size much greater than they ever had previously; this renewed growth then appeared in the muscles of the forearm, and they have continued to grow at a slow rate ever since. In the other case (Barth), an adult, in whom the disease began with pain in the lower limbs, the gastrocnemii were found to be below size; one year later hypertrophy was evident in many of the muscles formerly atrophied.

Charcot thought that a muscle he was examining in the early stage was smaller than normal. In Russell's case, mentioned above, of a child whose brother was affected with this disease, although the muscles of the calves were not enlarged, they were firm and hard when contracted, while the muscles of the upper extremities were extremely attenuated. May not this case belong to the same class as Pepper's?

Almost every muscle in the body may undergo this hypertrophic change, but the gastrocnemii are generally found enlarged; in the eighty-five cases there are only two exceptions, a case reported by Benedikt ("Elektrotherapie"), and Russell's case; next, the glutei are mentioned in forty-one cases; the extensor muscles of the spine in twenty-six cases; of the muscles of the upper extremities the deltoid and scapular are most frequently found involved in this change. The temporals are mentioned in three cases, masseter in two; the tongue was hypertrophied in three cases; four cases of hypertrophy of the heart were found; but as a rule the abdominal and thoracic viscera escape any change.

That the heart may become enlarged in connection with this disease, seems proved by the fact that in Foster's case, when the patient first came under observation, the heart-sounds were normal, and there was no hypertrophy; but, three years later, marked changes were found both in its size and sounds; there had been no disease to account for these changes.

All the muscles of the body may be increased in size (Du-

chenne, *loc. cit.*); the change may be confined to the four extremities (Coste and Gioja); may involve a muscle on one side of the body and not on the other, as the left latissimus dorsi (Benedikt); one gastrocnemius may be enlarged, the other atrophied (Müller, "Beiträge zur pathol. Anat. des mensch. Rückenmarks," Leipsic, 1871); may involve half of a muscle, as one-half of the deltoid (Benedikt); may attack the muscles on the posterior aspect of the leg, not involve those of the thigh, pelvis, or back, but may attack these of the forearm. In fact, there seems to be no rule as to its mode of distribution, and any attempt at classification would simply be an enumeration of the muscles involved in each case. In fifteen cases the calves only were enlarged.

While this increase is going on in some muscles, there is at the same time, or at some period of the disease, a wasting or atrophy of other muscles, adding much to the grotesque appearance of the patient. We often find that, while some of the muscles on the posterior part of the leg are hypertrophied, those of the thigh are atrophied; while those of the buttocks, again, are hypertrophied.

In nine cases the calves and forearms were increased in size, while the muscles of the thighs and arms were diminished; in five cases the lower extremities were reported enlarged, while the upper were wasted. In no case do I find the flexors of the foot reported enlarged.

When the muscles of the calves are enlarged, there is often found in the latter stage of the disease a condition resembling talipes equinus, with (in some cases) a tendency to varus—the foot being extended at the ankle-joint and not at the medio-tarsal joint, as in pure talipes equinus. This condition is reported in thirty-nine of the eighty-five cases; in four it is stated not to have existed; and in forty-two no mention is made of it. It is probably in the majority of cases a late symptom, yet in one case (Russell) it was found among the earliest; in one case, six months after the difficulty in walking was first noticed; in two cases, one year later.

In those cases in which it did not exist, in one the gastrocnemii had been hypertrophied only one year; in another

the calves had been enlarged five years, and in another three years.

Accompanying this condition of the feet we often find a claw-like bending of the toes, causing the patient to walk on the anterior portion of his feet. The cause of these deformities of the feet is attributed by most writers to the excessive action of the extensor over the flexor muscles of the foot. In my own case, when the patient first starts out to walk, she is able to bring her heels nearly flat on the floor; but, after taking one or two steps, the gastrocnemii are seen to contract more and more powerfully, almost spasmodically, and the heel is raised more and more from the ground, so that the weight of her body is thrown on the anterior portion of the foot; together with this there is a hurried gait, which ends by the patient falling down unless she supports herself by catching some firm object. There is some flexion of the leg on the thigh. T. Butlin ("St. Bartholomew's Hospital Reports," vol. viii., p. 194, 1872) considers that the hypertrophied muscles are shortened, not tonically contracted, but mechanically prevented from falling into their normal condition.

Passive motion at the ankle-joint in many cases can be made in all directions without exerting much force, but active motion is impossible. There is reported in thirty-six of the eighty-five cases a change in the cutaneous circulation, confined generally to the extremities, and although it is not mentioned by Duchenne, yet the number of cases in which it was found, show it to be not an uncommon if not quite a constant symptom. The change referred to is a mottling of the skin over certain portions of the body. In some cases it is of a bright-red color, in others it is at first red, but on exposure soon becomes bluish, while in other cases the skin is always of a dusky hue. In one case this mottling passed off when the patient assumed a horizontal position (Sigmundt, *Arch. für klin. Med.*, vol. vi., p. 630, 1866); in some cases this mottling is increased by muscular action. In all these cases there seems to be an inability to resist the chilling effect of the air, as exposure almost always deepens the shade of the marbling. In the case that forms the basis of this paper, on the slightest exposure the feet and legs assume an almost

leaden color, and even when in bed the lower, as well as the upper extremities, are of a dusky-marbled appearance.

In twenty-two cases this mottling was confined to the lower limbs; in eleven to both upper and lower extremities; in one case the whole body, except the face, was mottled.

In a case reported by Benedikt, the right side of the face was redder and sweat more than the left, the right pupil was dilated, and the sympathetic on that side of the neck was tender on pressure.

In one case (Drake) the skin is reported as assuming in patches a dirty-brownish discoloration, giving the surface a marbled appearance; the patches seemed to appear over those muscles which were about to undergo increase in size. The cause of this condition in the cutaneous circulation has been variously accounted for by writers, those who look upon pseudo-hypertrophic paralysis as a neurotic disease ascribing it to changes in the spinal cord, or the sympathetic, while others, among whom is Friedreich, refer the mottling to impeded circulation from obliteration of the veins in the hypertrophied muscles.

One observer points out the fact that the discoloration becomes more marked when the patient makes attempts to produce movements. The condition of the skin above mentioned is almost always associated with diminished temperature of the part; but in one case (Ord[1]) the calves which were hypertrophied were from 1.9° to 3.9° warmer than the thighs which were atrophied. The skin in this case was of a bright-red color.

In another case (Foster) the temperature of the limbs was higher when the mottling of the skin was brightest. This mottling seems to increase as the disease advances, and to become of a darker hue.

Nearly all observers, when they have made any mention of the electro-muscular contractility of the hypertrophied muscles, report that in the later stage of the disease it is diminished. Thus, in forty-eight cases, in sixteen it was normal; in fifteen diminished in both hypertrophied and atrophied muscles; in two it was normal in the enlarged, but diminished in the

[1] *Medical Times and Gazette*, November 15, 1873.

wasted; in four it was diminished only in those muscles that were increased in size; in three cases it was abolished in all muscles; and in two it was abolished only in those atrophied; in four it was diminished in the atrophied muscle only; in one exaggerated in the hypertrophied; in one case normal in the enlarged, abolished in the atrophied.

In the case of which I have now charge, there is certainly no diminution in the electro-muscular contractility to the galvanic current, except in the muscles on the anterior aspect of the leg. In a case reported by Foster, in which all the muscles, even those formerly enlarged, had undergone marked atrophy, so that the patient was almost an inert mass, being able to perform only a few movements, there was yet some response in all the muscles to the galvanic current, but varying, being greatest in those muscles which were least wasted. The pathological changes in the muscle would account for their diminished action to the galvanic current, being in progressive muscular atrophy in direct proportion to the amount of healthy muscular tissue, and inverse to the amount of fatty infiltration.

In the early stage of the disease, before any or but slight changes have taken place, the reaction is perfectly normal, or may even be increased; but later, after the muscular fibres have become embedded in fat, there would naturally be a weak response to even a very powerful current.

In regard to the electro-muscular sensibility, there do not seem to be any reliable data to form the basis of any analysis, on account of the majority of the patients being children, from whom no reliable information could be obtained, added to a mental weakness, as in my own case.

Duchenne mentions small, hernia-like protrusions in the hypertrophied muscles in several of his cases, but I fail to find them in the patient under my care, nor do I find any mention made of this condition by any other writer.

In some cases there is a marked hereditary character to the disease. Thus:

In two cases a maternal uncle and aunt had this disease.

In one case three maternal uncles and aunts had this disease.

In one case one maternal uncle and one half-uncle had this disease.

In one case three maternal half-brothers had this disease.

In one case a maternal half-brother, three maternal uncles, and other members on the mother's side, had shown the symptoms of pseudo-hypertrophic paralysis.

In thirty-seven instances, two or more belonged to the same family. It will be observed that it is only on the mother's side that this hereditary influence is transmitted; while the disease shows itself almost exclusively in the males. Thus in a case reported by Duchenne, the mother, while she escaped, transmitted the disease to the children of her marriage. The same fact is stated in Foster's case.

In one case a maternal grandfather was hemiplegic.

In one case a paternal grandfather was insane.

In one case a father was insane.

In one case a father was intemperate.

In one case two brothers died of granular meningitis.

In one case a brother was an idiot.

In fifteen cases of the eighty-five the family history was good.

In thirty-three cases no mention of family history is made.

The mental condition is mentioned in fifty-nine cases.

In twenty-eight it was good; in two it was idiotic; in twenty it was dull or weak; in one it was infantile at eleven years; in two it was fair; in four it was precocious; one patient was insane.

In twenty-six cases no mention is made of the mental condition.

In three of the cases where the mental condition was impaired, it came on after the development of the hypertrophy of the muscles. Two are reported by Kesteven.[1] In one the disease came on after an injury at fifteen, and at twenty-one his mental powers were slightly impaired. The other was bright and well until his sixth year, when his calves began to increase in size; at ten years he showed want of mental vigor, and at twelve, had epileptic convulsions. In a case reported by Estrazulas, (in the American supplement to the *Ob-*

[1] *Journal of Mental Science*, vol. xvi., April, 1870, p. 41.

stetrical Journal of Great Britain, etc., September, 1873), the patient was bright at his fifth year. The disease began at that date; and at the date of observation, when fourteen, his mental powers were weak.

In the case under my care the mental powers are weak, but her mother states that she was bright as a child.

In three cases epilepsy is mentioned as occurring after the disease had existed for some time; one became an epileptic at forty, a few years after the appearance of hypertrophic change.

One (Pepper[1]) at twenty began after the beginning of the disease. One (Kesteven[2]) after the disease had existed nine years. One patient (Hutchinson) had always been subject to fainting-fits.

In all of these the family history was good.

In ten cases the ophthalmoscopic examination of the eyes is reported. In only two was any change found: in one (Brown[3]) the vessels of the optic disk were small, and the papillary margin showed pigment maceration; in the other, there was some atrophy of the optic disk.

In quite a number of cases there seems to be a period in the disease during which there is neither an increase in the hypertrophy nor a diminution in the power of locomotion, before the final stage of the disease—atrophy—sets in.

This stage is mentioned only in ten cases, but from looking over the histories of all the cases there seems to be a time, in some cases longer, in others shorter, during which there is a pause in the advance of the disease. The shortest time mentioned was two years, and the longest ten years. In one case (Brunische[4]) this period, although not stated, evidently lasted eighteen years, and in a case (reported by Lutz[5]) there seems to have been a period of equal length.

In a few cases fibrillar contractions are reported to have beenn oticed (Wagner,[6] Griesinger,[7] Eulenburg,[8] Lutz,[9] Roth[10]).

[1] *Loc. cit.* [2] *Loc. cit.* [3] *Loc. cit.* [4] *Loc. cit.*
[5] *Arch. f. klin. Med.*, iii., 4, p. 358, 1867.
[6] *Berlin. klin. Wochenschrift*, No. 18, 1866. [7] *Loc. cit.*
[8] *Berlin. klin. Wochenschrift*, No. 50, 1865.
[9] *Arch. f. klin. Med.*, iii., 4, 1867, p. 358. [10] *Arch. d. Heilk.*, 1871, ii.

Friedreich[1] thinks that they always exist in the diseased muscles, but that they escape notice on account of the fat with which they are covered; but most observers consider that it is a very rare symptom.

Formications are mentioned in one case (Roth),[2] and numbness of the hands and feet in one (Russell).[3]

In four cases speech was slow and indistinct (Duchenne, Barth, Roth, Dahlerup); in two of these deglutition was also difficult.

The functions of the bladder and rectum are not involved, and, in those females who were old enough, menstruation was perfectly normal.

Sensation, as a rule, is normal, but in one case it was blunted all over the body (Roquette), in one diminished below the loins (Kesteven), and one is reported as being diminished in many places (Benedikt).

A period at length arrives in the course of the disease in which all the paralytic symptoms are aggravated, in which the atrophy is more marked in those muscles which have already undergone wasting, and sometimes even in those which have been hypertrophied. The date of this period is as uncertain as that of the other stages; it may be postponed for many years, or it may follow soon upon the other changes which have been mentioned. But as in progressive muscular atrophy, so in pseudo-hypertrophic paralysis, the progress of the disease, when once well established, is onward toward certain death. These patients gradually lose all power over their muscles, and are often reduced to mere skeletons. The longest time that one affected with this disease is reported to have lived is thirty years (Müller). The shortest is two years (Webber[4]). Between these two extremes there is every degree of variation. It is a very chronic trouble, and seldom runs its course in less than six years.

In nineteen cases, the time from the first manifestation of the disease until the paralyses became so great that the patients were unable to stand, was as follows:

[1] *Ueber Progressive Muskelatrophie*, Berlin, 1873.
[2] "Constatt. Jahr.," 1867, 2, 1, p. 294. [3] *Loc. cit.*
[4] *Boston Medical and Surgical Journal*, November 17, 1870.

1 case	5 years.	7 cases	9 years.
1 "	6 "	4 "	11 "
3 cases	7 "	1 case	12 "
1 case	8 "	1 "	22 "

In a few cases flexions of the joints are mentioned as occurring late in the disease (Wagner, Estrazulas, Clark). In one case the ankle, knee, hip, and elbow joints were strongly flexed and rigid.

The termination of the disease is given in thirteen cases:

Two died of phthisis: one at ten, in the eighth year of the disease; one at fifteen, in the twelfth year of the disease.

Three died of pleuro-pneumonia: one at fourteen, in the twelfth year of the disease; one at nineteen, in the twelfth year of the disease; one at thirteen, in the eighth year of the disease.

One, of a low form of pneumonia in his fourteenth year—eleventh of disease.

Three of pneumonia: one at five, in the second year of the disease; one at forty-three, in the seventh year of the disease; one at thirty-four, in the thirtieth year of the disease.

Two of bronchitis: one at fourteen, in the tenth year of the disease; one at about twelve years.

One from hypertrophy of the heart, at eighteen.

One, from croup, at nineteen, in the fourteenth year of the disease.

The pathological anatomy of the disease may be considered under two heads, namely, that of the muscles themselves, and that of the nervous centres.

First, in regard to the muscles.

In my own case, I removed, last December, small pieces of the muscular tissue from the left gastrocnemius, sacro-lumbalis, left deltoid, left biceps brachii, tibialis anticus, and left vastus externus. The gastrocnemius, sacro-lumbalis, and deltoid, were hard, and the rest were soft and flabby, but I do not think atrophied, except it was the tibialis anticus.

The muscles were examined when fresh. The following is the report of Dr. Bull, who made the examination for me: Gastrocnemius, muscular fibres of normal size, but with irregularly serrated, or rather roughly-scalloped edges. Little or no

connective tissue between the individual fibrillæ, but bundles of fibrils are surrounded by very-much-hypertrophied connective tissue, the transverse striation in most of them still plain, the longitudinal striation very distinct; in some cases very distinct nuclei present.

In most of the connective-tissue fibres, more or less fat-globules, which were so numerous in many fibres as to constitute an actual fatty degeneration.

Deltoid.—The same relative arrangement and increase of connective tissue. The muscular fibres are notched, or scalloped, as in the gastrocnemius. Transverse striation very distinct. Longitudinal striation less distinct than in the gastrocnemius, very little fatty deposit in or between the fibres. Muscular fibres have a general homogeneous, almost structureless appearance.

Biceps Brachii.—Same arrangement of connective tissue. Transverse and longitudinal striation tolerably well marked, scarcely any fatty deposit, and in some places fibres have a structureless appearance like empty sarcolemma.

Tibialis Anticus.—Connective tissue scarcely present at all, muscular fibre very little notched, transverse striation faint, longitudinal striation very plain. Quite a considerable deposit of fat-globules in and between the muscular fibres.

From numerous microscopic examinations of the muscular tissue in pseudo-hypertrophic paralysis, certain pathological changes are found common to all. The earliest change noticed is in the delicate interstitial connective tissue, which in the normal state scarcely separates the primitive muscular fibres from one another; this is increased in quantity, and in time is replaced by thicker bands, the diameter of which at certain points equals, or even exceeds, that of the muscular fibres themselves (Charcot)[1]. This tissue is of recent formation; in the earliest period of the disease the fibres are studded with nuclei, embryoplastic or fusiform cells; but in those muscles in which the change is more advanced, the nuclei and cells are diminished in numbers, and the bundles are formed exclusively of fasciculi of long, undulating fibres (Charcot). While these changes are going on in the interfibrillar connective tis-

[1] *Archives de Physiologie*, March, 1872.

sue, the larger quantities of the same tissue between the muscular bundles undergo correspondingly larger increase (Butlin[1]). Section made in various directions shows that this increase is between the muscular fibres, separating them from one another, often to a considerable distance (Butlin).

After this hyperplasia has gone on for some time, another change is noticed: there are seen fatty cells, at first scattered here and there between the bundles of connective tissue; these increase in number and coalesce, forming for themselves interspaces or meshes between the newly-formed fibrous tissue, the deposit being more abundant where the connective tissue is in greatest quantity (Butlin), so that at last it distends more and more the cellular elements, which gradually disappear, so that the muscle consists mainly of fatty tissue,[2] that is, in those muscles that are enlarged (Charcot, Butlin, Clark, Duchenne). In those muscles which do not undergo hypertrophy, but on the other hand atrophy, examinations do not agree; in one case where the deltoid was wasted (Pepper), the only difference between it and the gastrocnemius, which was enlarged, was that in the former there was less fat found; in another case (Estrazulas) there was no increase in the interstitial connective tissue, nor any fat. In one case (Roth) the subcutaneous fat was greatly increased in quantity and dipped down between the individual muscles. When the fatty substitution has reached its utmost limits, the appearance of the muscle on section to the naked eye is that of fatty tissue; all muscular structure seems to have disappeared (Charcot), and microscopic examination of such a muscle shows it to consist of distended fatty cells and a considerable amount of connective tissue (Clark). But Charcot states that he found some muscular fibres perfectly normal in a muscle that on inspection appeared to consist entirely of fat.

While these changes are going on in the connective tissue, some of the primitive muscular fibres become altered in appearance, while others, on the other hand, undergo but little, if any, change. The earliest alteration noticed is in their

[1] *Loc. cit.*

[2] In Russell's case there was no fatty infiltration; the change consisted only of a hyperplasia of the connective tissue.

color; they become pale, and the transverse striation becomes faint, or entirely disappears, while the longitudinal markings become more distinct (Duchenne, Clark, and others); later, in some fibres both the longitudinal and transverse striation have disappeared, and the fibres present a hyaline or ground-glass appearance (Charcot, Pepper). Many fibres, later on in the disease, undergo atrophy, especially in their diameters, some being reduced to one-quarter, while with others it is necessary to use the greatest care to distinguish them from the connective tissue (Charcot). Some observers (Cohnheim, Butlin, Russell) mention fragments of greater or less length with distinct outlines, which were looked upon as empty sarcolemma-sheaths. Clark found the same appearance, and, on carefully following them up for a little distance, transverse striation showed itself, at first faint, then more distinct, until the aspect of distinct fibres was assumed.

The muscular fibres do not undergo any fatty degeneration; almost all observers state distinctly that they fail to find any fat-cell within the sarcolemma-sheath in the majority of fibres. Roth states that a few fibres exhibited this change.

Some fibres, and these of small diameter, present an appearance as though the sarcos element was finely divided, giving them a granular appearance (Charcot, Clark).

In two cases the muscular fibres were larger than normal (Pepper, Lyden). Pepper considers that they may temporarily share in the exaggerated nutrition; he found some of the fibres of the gastrocnemius one-third wider than normal.

Cohnheim mentions a division or splitting of the muscular fibres into two or even three sections of equal width.

Roth reports a swollen condition of the muscular fibres in an enlarged gastrocnemius.

In one case (Clark) some of the muscular fibres were narrower at one place than at another, but I do not find any reference to a notched or scalloped appearance, as in my own case.

These changes in the fibres take place earlier in some than in others; so that there are often found those which have undergone atrophy, side by side with those that are perfectly normal, or are even increased in size (Eulenburg, Knoll,[1] Müller); these

[1] "Wien. med. Jahrb.," i., 1872.

changes have been looked upon by some as compensatory in their character. The change, then, in the muscular fibres would seem to be a gradual, simple, progressive atrophy, not accompanied by any fatty degeneration such as is found in progressive muscular atrophy, preceded by a hyperplasia of the connective tissue.

But six *post-mortem* examinations have been recorded in which the nervous centres were carefully examined, namely by Cohnheim, Barth, Kesteven, Charcot, J. Lockhart Clark, and Müller.

In 1865 Cohnheim examined the cord and nerves, including the sympathetic, in a case in which the disease had reached an advanced stage, and found them all perfectly healthy ("Constatt. Jahrb.," 1866, ii., p. 261).

Kesteven, in a patient who had been for some years an epileptic, with failing mental powers, so that at the time of his death he was an imbecile, and in whom the disease had passed into its last stage, found the perivascular canals dilated and circumscribed spots of granular degeneration scattered through both brain and spinal cord. The *cells* of the *gray* matter both in the brain and cord retained their normal character (*Journal of Mental Science*, April, 1870).

Barth, in a man forty-three years old, who exhibited symptoms not usually found in those suffering from pseudo-hypertrophic paralysis, found the spinal canal lined with a layer of fatty tissue, so that the dura mater was crowded very far inward; the intervertebral substance was infiltrated with fat, the ganglia were surrounded by fat, which extended through the intervertebral foramen. The spinal cord was of normal thickness. After being hardened in chromic acid, and colored with carmine, on sections being made, there was found gelatinous degeneration of the neuroglia in the anterior gray matter and lateral columns, degeneration of the nerve-fibre, and in their place was found a granular mass with numerous corpora amylacea. As far as the cervical portion was concerned, the changes had occurred more in the lateral columns, while in the dorsal region the anterior were also affected; in the lumbar portion the alteration was more marked than in the cervical (*Arch. d. Heilk.*, 1871).

Müller, in a woman twenty-four years old, the subject of dementia, reports disease of the brain—gray degeneration of the lateral columns. There was also an excess of fatty cells in the cellular tissue around the spinal cord, and fatty deposits in the cord itself ("Beiträge zur Anat. des mensch. Rückenmarks"). The interstitial connective tissue in the tibial, sciatic, and perineal nerves, was increased in quantity and infiltrated with fat.

Charcot (*Archives de Physiologie*, March, 1872) reports the examination in a case where the disease had existed ten years. Sections were made from different regions of the cord, and also from the sciatic, radial, and median nerves; these were hardened in chromic acid, colored with carmine, and prepared with great care. The result of the examination was absolutely negative. Everywhere the white antero-lateral and posterior columns were in a state of perfect integrity. The gray substance, which was made the object of special study, did not present any trace of alteration. The anterior horns were neither atrophied nor deformed; the neuroglia had its normal appearance; the motor cells, in normal numbers, did not show, in their different parts, any deviation from the physiological type. The spinal roots, anterior as well as posterior, appeared equally healthy. Examination of the sciatic, median, and radial nerves showed that they were perfectly healthy.

Within the past year Dr. J. Lockhart Clark[1] has reported the result of an examination of this disease in a boy, whose muscles were in the most advanced stage, that of atrophy, not only of those muscles not hypertrophied, but even of those that had at one time shown this condition in a marked degree. There was no affection of the mind; the sphincters acted well.

"The brain, medulla oblongata, and meninges of the cord, were healthy. The spinal cord presented various changes throughout the cervical, dorsal, and lumbar region. The most important was disintegration of the gray substance of the anterior, lower, and central portions of each lateral half. In some places this had occurred chiefly around the vessels, but in others it involved extensive areas; about the level of the

[1] *Loc. cit.*

last dorsal nerves it had amounted to almost total destruction of gray matter on each side, between the posterior vesicular columns. Other changes, as disintegration of the nerve-roots, commencing sclerosis of the lateral and posterior columns, destruction of the white commissure in various places, dilatation of vessels, and extravasations, were noticed.—("Medico-Chir. Trans.," 1874.)

In looking over the results of the examination in these six cases, it is evident that the alterations found are very diverse, and seem to deny that there is found in the spinal cord any lesion common to all cases.

Müller's and Kesteven's cases must be discarded, as the concomitant diseases, paralytic dementia, and imbecility with epilepsy, would throw doubt on the pathological signification of the lesion found, being similar to changes described by writers on these diseases.

Barth's case is not open to as weighty objections, although the acute pain in the limbs, its initial symptoms, the ptosis, as well as other groups of symptoms not found in pseudo-hypertrophic paralysis, might justly raise the question as to its identity with the disease under consideration.

Barth reports disintegration of the nerve-fibres in the gray matter of the lateral column.

Clark found similar changes, together with disintegration of the nerve-roots, sclerosis of the posterior columns, and lesions found associated with other diseases, in which there is no clinical history of hypertrophy. The result of the careful examination of Charcot seems to prove the fact that the disease may run its course without any appreciable lesion of the cord, or peripheral nerves, and that the changes found in the spinal cord are not necessarily connected with the change in the muscles.

The supposed exciting cause is reported in a few of the cases. In three the disease seemed to come on after an attack of measles (Stoffila, Griesinger); and in another (Uhde) the disease made more rapid progress after an attack of rubeola. In two cases the disease is ascribed to cold and damp rooms, one to sitting on the damp ground; two had chronic diarrhœa in infancy.

A case is reported by Kesteven of a boy aged seventeen years, healthy and well-formed, with a good family history, who, when he was fifteen years of age, fell backward from a chaise, striking on his back. He suffered no ill effects from the accident, beyond a severe pain extending around his body on the level of the umbilicus; this, however, soon passed away. Some time after, difficulty in walking was experienced, followed by the other characteristic symptoms of pseudo-hypertrophic paralysis.

In another case (Müller) the disease is ascribed to a fall from bed.

Racowac mentions a rapid increase in the symptoms after an acute disease.

But little can be said in regard to treatment. In seventeen cases, two are reported cured by Duchenne faradization, aided by massage and hydropathy; both of these patients were in the early stage of the disease; treatment extended over the space of six months.

Three are reported by Benedikt as improved, one by faradization, and two by galvanization of the sympathetic. One of these suffered a relapse in four months, but improved again on resuming the galvanic treatment.

Eight patients derived no benefit from faradization, four no benefit from any kind of treatment.

In the case reported by Uhde (*Arch. klin. Chir.*, 1874, p. 517), of a boy eleven years old, in whom the disease had existed five or six years, whose gastrocnemii were enormously enlarged, as well as other muscles of the lower portion of his body, while the upper extremities were atrophied, and who was unable to stand, gymnastics and faradization were employed. On October 29th tenotomy of the tendo-Achillis on both sides was done, together with that of the plantar fascia. One month later he could get out of bed and stand, and by December 25th could walk with support. About the middle of January he could go up-stairs without aid.

In one of the cases reported by Meryon, no benefit was gained by tenotomy and orthopædic treatment, under supervision of Mr. Tamplin. Mr. Adams obtained no permanent benefit from similar treatment.

It remains, in conclusion, to mention the different theories advanced by pathologists in regard to this disease. Duchenne is inclined to think that the paralysis is due to a formative irritation, which produces proliferation of the connective tissue in the muscles, but is at a loss to explain the cause of this irritation.

Colnheim considers the disease a general lypomatosis.

Charcot states that it is not due to any appreciable change in the spinal cord, but is inclined to attribute it to some lesion of the sympathetic; while Dr. J. L. Clark is satisfied that the changes in the cord, which he has described, are connected with those found in the muscles as their cause. Kesteven advocates the same views.

Friedreich, in his work above referred to, in which he devotes considerable space to the consideration of pseudo-hypertrophic paralysis, considers it as identical with progressive muscular atrophy, and looks upon the enlargement of some muscles as an accidental process in the course of the latter disease.

He considers that "in both diseases the cause of the atrophy begins within the muscular tissue, as an active inflammatory process, which in its histological characteristics corresponds with other forms of chronic myositis. Sooner or later the inflammatory irritation communicates itself to the intra-muscular nerves, which further is propagated to the nerve-trunks, and thus to the cord;" and that the morbid process may come to a stand-still at any stage of its course. He in this way reconciles the contradictory results of the examination of the cord. But he fails to assign any cause for this inflammation of the muscular tissue.

If the morbid process extends from the muscles to the nerves by contiguity of tissue, and thus to the cord, why do we so seldom find the functions of the nerves of sensation altered?

If the views of Vulpian, Bidder, and Waller, are correct, that the fibres connecting the two great divisions of the nervous system pass both ways to and from the cord to the ganglia of the sympathetic, and that those fibres passing from the ganglia to the cord are for the purpose of supplying

it (the cord) with trophic nerves, while those going to the muscles from the same ganglia preside over their nutrition, what might be the effect of a morbid change taking place in the ganglia? Might it not in one case involve those cells which preside over the nutrition of the muscles, and in another case involve those going to the cord as well?

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